OPERATION IN SPINAL-CORD INJURIES.

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IN lesions concealed from the surface involving a central nerve organization of extreme delicacy, the extent and precise nature of which are unknown without exploration, the after effects of such far-reaching importance, the parts reasonably accessible, and the possibilities so great of benefiting the patient, the operative stand-point in injuries to the spinal cord is found to be of special interest.

This paper is based upon two cases of spinal-cord injury which Dr. Samuel J. Mixter permits me to report. Both cases were admitted upon Dr. Mixter's service at the Massachusetts General Hospital, Case I coming under my care while Surgical House Officer.

In literature there are numerous valuable articles on injuries of the spinal cord by Thomas,[1] Courtney,[2] Thorburn,[3] Walton,[4] Kocher,[5] Bastian,[6] Lloyd,[7] Löwenthal,[8] Taylor,[9] and others. Of recent papers that of Walton emphasizes the value of operation.

Case I demands interest and attention for the following reasons: 1st, Because there is high fracture of the spine, viz., in the cervical region; 2d, It was operated upon within twenty-four hours; 3d, Unusual length of life following the operation; 4th, Marked improvement in motion and sensation; 5th, Following death there was preserved a complete series of sections of the cord from the pons to the coccyx, making it possible to reproduce sections at different levels showing degeneration, which I have been unable to find in previous literature; 6th, Its bearing upon the question of early operation, and the fact that

if clinical observation suggests total injury of the cord, an operation of little danger in itself, may relieve the pressure and allow the continuation of normal function of uninjured fibres, which would otherwise succumb to pressure and degeneration.
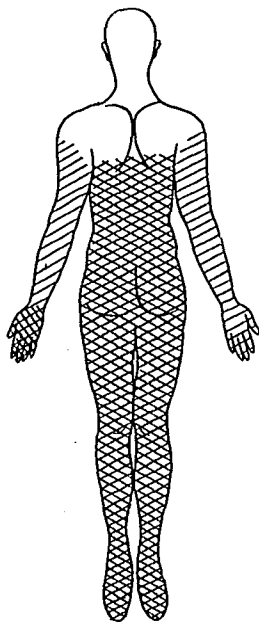
CASE I.—On the morning of June 24, 1902, a man forty years of age was brought to the accident room of the Massachusetts

FIG. 1.   FIG. 2.
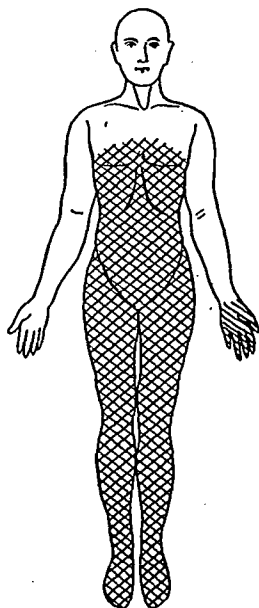
Front.   Back.
/ / / / = Absence of motion.   \ \ \ \ = Absence of tactile sense.

General Hospital with the history of having fallen eight feet from a tree, striking on the back of his neck. The injury occurred at 7.30 P.M. the previous evening. It was said that he did not lose consciousness.

Upon admission to the hospital he was conscious; pupils reacted to light and with accommodation; he was unable to move

his head. Hearing and sight were normal; throat examination was negative. He complained of headache and pain in the neck. There were no external wounds. There was diaphragmatic breathing, complete loss of tactile sensation, flaccid paralysis below level of fourth rib in front, and below a line from the fourth to the seventh rib in the axillary line (Figs. 1 and 2), loss of reflexes. No broken bones were palpable, and no deformity of spine detected. Motions of shoulders slow; arms could be drawn up to chest slowly, but not extended. Unable to put either hand on the opposite shoulder. Type of sixth cervical nerve irritation.

Seen in consultation with Drs. Walton, Baldwin, and Paul, who considered the lesion to be in the region of the fifth and sixth cervical vertebræ.

Operation by Dr. Samuel J. Mixter; Dr. H. M. Chase assisting.

Patient on abdomen, head flexed over the end of the table; incision in median line of neck, spinous processes exposed and laminæ and spines of fourth, fifth, and sixth cervical vertebræ found fractured, depressed, and apparently lying against the cord. These were removed, exposing the dura, which looked normal. The dura was opened and cerebrospinal fluid escaped, no blood-clot under dura, small clot found under laminæ. Dura not sutured, wick inserted, superficial sutures, and bandage. Good recovery from ether. Temperature gradually rose to 104.6° F.; pulse slow and good volume.

June 25, 1902. No change in tactile sensation, arms are moved a little more deliberately.

June 26, 1902. Tactile sensation returning slightly.

June 30, 1902. Tactile sensation recovered over whole body except left hand (Figs. 3 and 4). Can locate accurately where one touches him, though sensation is not normal, as shown by the time required to locate the point of contact. Plantar reflexes, knee-jerks, and ankle clonus absent. Slight thoracic breathing. "Soreness" of muscles of arms. Paralysis still complete below dotted line, and of extensors of arms.

July 6, 1902. Slight plantar reflex and knee-jerk. No cremasteric reflex. Improvement in motion of left arm.

July 15, 1902. Ankle clonus present, tactile sensation normal except in left hand.

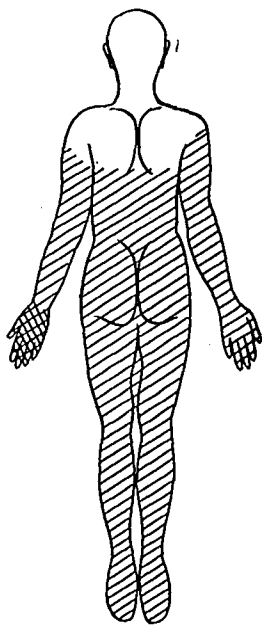July 26, 1902. Hamstring muscles can be contracted, but not

strong enough to raise the leg. Right foot can raise the great toe and next two toes. Left foot can move the great toe occasionally. Active knee-jerks.

FIG. 3.   FIG. 4.
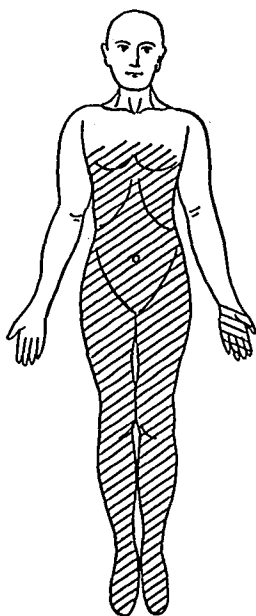
Front.   Back.
/ / / / = Absence of motion.

August 4, 1902. Can feebly flex and extend both forearms, slightly flex the fingers of right hand, occasionally raise both knees, and move toes of right foot.

August 22, 1902. Tactile sensation preserved throughout, except ulnar side of left hand, front and back. No motion in left leg. Right thigh and toes move voluntarily. Areas of temperature sense, pain sense, and hyperæsthesia as in Figs. 5 and 6.

September 10, 1902. Marked atrophy of muscles. Abdomen periodically distended, relieved by enemas.

September 21, 1902. Considerable pain and twitching in muscles of abdomen, arms, and legs.
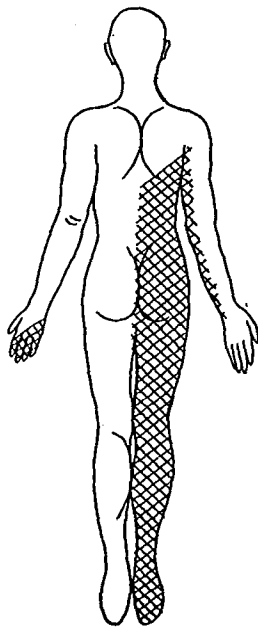
October 1, 1902. Right arm; can use muscles of shoulder, biceps, triceps, extensors of fingers and wrists, and very slight motion in flexion of fingers. Left arm considerably weaker than right. Can use muscles of shoulder biceps and muscles of forearm.

FIG. 5.   FIG. 6.
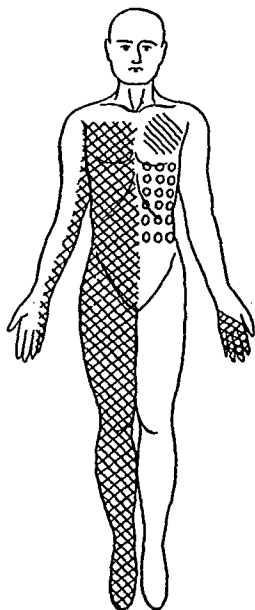
Front.   Back.
/ / / /= Temperature sense.   \ \ \ \= Pain sense.   o o o o = Hyperæthesia.

October 10, 1902. Right leg; can flex, extend, and adduct right thigh; can flex and extend knee fairly vigorously with weak action of muscles of calf and anterior tibial muscles. Left leg, very slight power in muscles of left thigh, no power in muscles of left leg.

October 20, 1902. Gaining slight control of muscles of back, especially on right.

November 15, 1902. Now lacks motion in muscles of respiration and abdomen. No cystitis and no bed-sores.

November 30, 1902. Gaining in every way; can sit up in chair without assistance; can move wheel-chair a little with right hand.

Disturbances of sensation mapped out in Figs. 5 and 6 on date of August 22, 1902, have nearly disappeared, and areas are blurred and indistinct.

December 12, 1902. Occasional gastric disturbance.

January 1, 1903. Can pass urine voluntarily, but cannot empty bladder. Good control of sphincter ani. Flexor muscles gaining faster than extensor muscles. Can feed himself with right hand.

January 17, 1903. Cannot hold anything in left hand.

February 18, 1903. Can use both hands to wheel himself around in chair. Urine examination finds color normal, acid, sp. gr. 1029, albumen slight trace, sugar absent. Rare hyaline cast with fatty renal cell adherent, considerable pus, few medium size and small round cells.

March 6, 1903. Chill, vomiting, temperature, epigastric pain, distention. No abdominal spasm. Lungs negative.

March 10, 1903. Chills, pain on left side of abdomen. Slight tenderness over left kidney. No plasmodia. White count, 9000.

April 1, 1903. Out in chair apparently in good condition; can hold book in both hands for reading. Moderate cystitis.

May 1, 1903. More improvement on right side than on left; can sit up and raise himself with his arms, but cannot stand.

May 14, 1903. Urine examination, pale, clear, alkaline, sp. gr. 1018, trace of albumen, sugar absent. Many triple phosphate crystals, much pus, considerable normal blood, rare caudate cell, several small and medium-sized round cells.

May 20, 1903. Chill, fever, pain on left side of abdomen. No tenderness over kidneys. White count, 14,000. Increased to 20,500 in three days.

May 21, 1903. Urine examination, color normal, acid, sp. gr. 1012, trace of albumen, sugar absent. Many hyaline and fine granular casts, few with renal cells and blood-corpuscles adherent, considerable pus.

May 25, 1903. Chills more frequent with pain in left side. More marked cystitis with renal complication.

May 30, 1903. Suddenly became much worse, chills, great weakness and difficulty in breathing. Weakened rapidly, and died June 5, 1903.

Autopsy.—June 6, *1903,* by Dr. Oscar Richardson.

Anatomical diagnosis: right nephrolithiasis, occluding stone in the right ureter; pyelonephritis; cystitis; hyperplasia of spleen; small infarct in middle lobe of right lung; dural adhesions in region of upper cervical portion of spinal cord.

On opening the spinal canal, the cord presents nothing remarkable until the upper cervical region is reached, where the dura is firmly adherent to the vertebræ and to the cord. Outwardly there are no deformities of the cord. Entire cord placed in hardening fluid.

Kidneys combined weight 440 grammes. Capsules strip leaving smooth surface. Pelvis of right kidney dilated, its mucosa grayish-red to black, bathed in semifluid, dirty yellow purulent material. Calices markedly dilated with concretions. Right ureter dilated, occluded by small yellowish stone one and one-half centimetres in greatest diameter. Two pockets containing stones at pelvis of kidney. Left kidney and ureter not remarkable. Bladder contains foul fluid.

I wish here to express my appreciation of the courtesies extended me by the Clinico-Pathological Laboratory of the Massachusetts General Hospital, and especially for the interest of Dr. Oscar Richardson, who performed the autopsy and removed the brain and spinal cord, placing same in hardening fluid, and later staining and mounting a series of eighty-five cross-sections from the pons to the coccygeal segments, which permitted me to reproduce characteristic sections of each region of the cord to illustrate the tracts of degeneration of nerve-fibres. The sections were stained by the W. Ford Robertson's [10] osmic acid method.

Cross-sections of the cord at seven levels have been selected, —one through the second lumbar segment, the sixth dorsal or thoracic segment, the eighth cervical segment below the lesion, one through the lesion at the sixth cervical segment, above the lesion at the third cervical segment, one through the oblongata, and through the pons.

With transverse lesion at any level of the spinal cord, one expects to find evidence of descending degeneration of motor

tracts below the lesion, of those axones which have been severed from their central cells. With this, also, is a short descending degeneration of some collaterals from sensory fibres. Ascending degeneration occurs above the lesion of such sensory fibres as have been severed from their nerve-cells in the dorsal root ganglia.

The following sections have been carefully compared with those pictured in Bruce's atlas, and the levels accurately established.[11]

PATHOLOGICAL ANATOMY.

*Right Half of Cord.**—Ventral root normal. Direct pyramidal tract shows degeneration of a few scattered fibres. Motor cells in ventral horn more numerous and more pigmented than on opposite side. Crossed pyramidal tract shows marked degeneration, with scattered normal nerve-fibres, which increase in number towards the lateral limiting area. Dorsal root not degenerated on either side. (Fig. 7.)

Left Half of Cord.—Ventral root shows degeneration with scattered normal nerve-fibres. Direct pyramidal tract slightly more degenerated than on the right side. Motor cells in ventral horn fewer and contain less pigment than on the right side. Crossed pyramidal tract shows larger area of degeneration with fewer scattered normal nerve-fibres.

General.—A few scattered fibres of degeneration of direct pyramidal tract are seen in the lumbar segments, but this tract cannot be followed to show degeneration in either sacral or coccygeal segments. No degeneration in dorsal columns (short sensory pathways affected by the lesion stopped at a higher level). Of the pyramidal tracts, the greatest degeneration is in the crossed. Cross pyramidal tracts diminish in lower segments. Collaterals, probably representing the reflex arc, are seen going from the dorsal to the ventral horn. These, as would be expected, do not show degeneration.

Right Half of Cord.—More marked degeneration of direct pyramidal tracts than on opposite side. Crossed pyramidal tracts show less marked degeneration than on opposite side. Clark's

* I wish to state at this point that by the words left and right half of the cord is meant from the reader's position viewing the diagram.
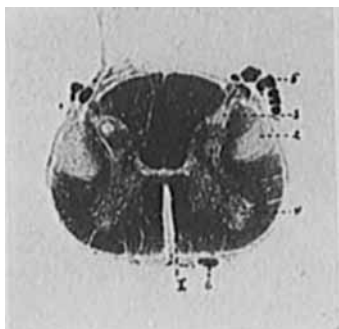
* FIG. 7.—Degeneration of motor (pyramidal) tracts. Second lumbar segment. 1. Direct pyramidal tract. 2. Crossed pyramidal tract. 3. Substantia gelatinosa of dorsal horn. 4. Ventral horn. 5. Dorsal root. 6. Ventral root.
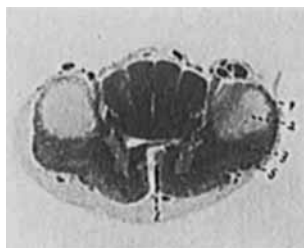

FIG. 8.—Degeneration of motor (pyramidal) tract. Sixth dorsal segment. 1. Direct pyramidal tract. 2. Crossed pyramidal. 3. Column of Clark. 4. Direct cerebellar tract. 5. Gower's tract.
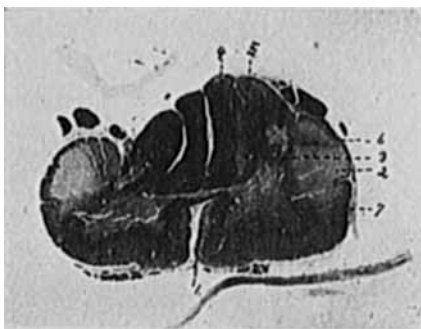
FIG. 9.—Degeneration of motor (pyramidal) tracts. Eighth cervical segment. 1. Direct pyramidal tract. 2. Crossed pyramidal tract. 3. Comma tract (Schultz's area). 4. Column of Goll. 5. Column of Burdach. 6. Direct cerebellar tract. 7. Gower's tract.
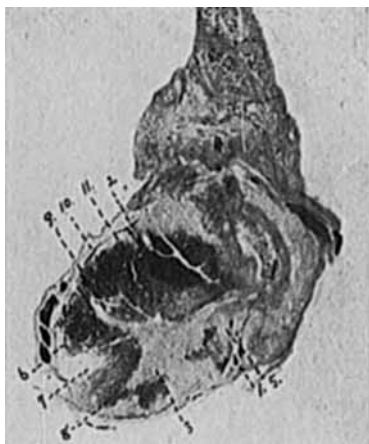

FIG. 10.—Section through the lesion. Sixth cervical segment. 1. Ventral fissure. 2. Dorsal fissure. 3. Ventral horn. 4. Fibrous tissue. 5. Direct pyramidal tract. 6. Direct cerebellar tract. 7. Crossed pyramidal tract. 8. Gower's tract. 9. Dorsal horn. 10. Column of Burdach. 11. Column of Goll.

column well defined on both sides. Direct cerebellar tract more defined on this side. (Fig. 8.)

Left Half of Cord.—Marked degeneration of direct pyramidal tracts slightly less than on right side. Motor cells show same changes as in previous section. Crossed pyramidal tract shows more complete degeneration than on the right side, though there are a few normal fibres.

General.—No degeneration in dorsal columns; this section is too low for degeneration of descending collaterals of sensory nerves affected by the lesion. Areas of all pyramidal tracts increase in higher levels. The inner side of Gowers's tract lies against the lateral ground bundle not sharply defined, as there is a considerable admixture of fibres.

Right Half of Cord.—Motor cells more numerous and more pigmented than on the left side. Direct pyramidal tract shows more degeneration than on left. Right dorsal root has small area of degeneration. Crossed pyramidal tracts show extensive degeneration. Comma tract at base of Burdach's column shows degeneration of short descending collaterals of sensory neurones. (Schultz area.). (Fig. 9.)

Left Half of Cord.—Crossed pyramidal tract shows most complete degeneration on this side. Direct cerebellar tract sharply defined.

General.—Distortion of the section probably artificial.

Right Half of Cord.—Adhesion of large mass of fibrous tissue. Great destruction of cord with irregular areas of degeneration and fissures caused by crush. The ventral and dorsal horns cannot be distinguished owing to their involvement in the lesion. Both motor and sensory tracts are degenerated, but not definitely outlined. Greatest degeneration around periphery. Dorsal root is degenerated. (Fig. 10.)

Left Half of Cord.—No mass of adhesion. Ventral horn quite definitely outlined, containing an occasional motor cell and small amount of pigment. Areas of degeneration more definitely outlined than on right side.

General.—This section, contrasted with those immediately above and below, shows the greatest amount of destruction. Destruction evidently greatest on right, though scattered normal fibres can be detected.

Right Half of Cord.—Ventral horns apparently normal, with

small amount of pigment in motor cells. Degeneration of
Gowers's and direct cerebellar tracts more marked on this side.
Marked degeneration of the columns of Goll and of Burdach.
(Fig. 11.)

Left Half of Cord.—Marked degeneration of dorsal columns.
Degeneration of Gowers's and direct cerebellar tracts.

General.—On each side the portion of Burdach's column
adjacent to the dorsal horn is most perfectly preserved (middle
root zone), showing that degeneration of the dorsal root was at a
lower level, and that these fibres take a position nearer the dorso-
median line as they ascend. Motor tracts do not show degenera-
tion above the lesion.

In this section only ascending degeneration of sensory tracts
is seen. The direct cerebellar tract shows more degeneration on
left side. Gowers's tract, above and a little external to the inferior
olivary nucleas and external to the nucleas lateralis, shows as a
small area of degeneration. (Fig. 12.)

A number of observers believe that a portion of Gowers's
tract continues directly or by relay into the Lemniscus mediales.
Flechsig follows the tract into the oblongata as far as the region
of the nucleas lateralis, where it lies close to the periphery (ap-
proximately the level of this section). Löwenthal carried the
investigation of its distribution into the cerebellum.

The columns of Goll and Burdach cease in the gracile and
cuneate nuclei respectively, therefore only those axones are degen-
erated in this section whose central cells lie at a higher level.

I am unable to detect degenerated fibres in this section. It is
possible that a few fibres of Gowers's tract have ascended to this
level and entered the Lemniscus medialis. This section is through
the superior cerebellar peduncle. (Fig. 13.)

Summary.—High fracture of spine, viz., sixth cervical
vertebra. There were present all the classical symptoms upon
which authorities have previously based their opinion that
operation was contraindicated because it suggested total trans-
verse lesion with a crush of the cord beyond repair. The case
was operated upon within twenty-four hours. The patient lived
eleven and a half months, during which time there was marked
and steady improvement; from a condition of total paralysis
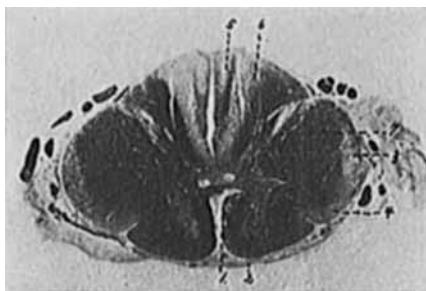over an area represented in Figs. 1 and 2, the patient regained

FIG. 11.—Degeneration of sensory tracts. Third cervical segment. 1. Direct pyramidal tract. 2. Ventral horn. 3. Direct cerebellar tract. 4. Gower's tract. 5. Column of Goll. 6. Column of Burdach. 7. Crossed pyramidal tract.
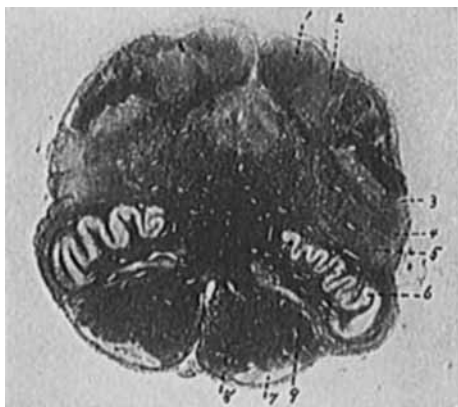
FIG. 12.—Degeneration of sensory tracts. Oblongata. 1. Nucleus gracilis. 2. Nucleus cuneatus. 3. Direct cerebellar tract. 4. Gower's tract. 5. Nucleus lateralis. 6. Inferior olivary nucleus. 7. Nucleus arcuatur. 8. Ventral pyramids. 9. Accessory olive.

FIG. 13.—Section through superior cerebellar peduncle. 1. Superior peduncles. 2. Posterior longitudinal fasciculus. 3. Fillet lateralis. 4. Lemniscus medialis. 5. Nucleus centralis superior 6. Transverse and longitudinal fibres of pons.

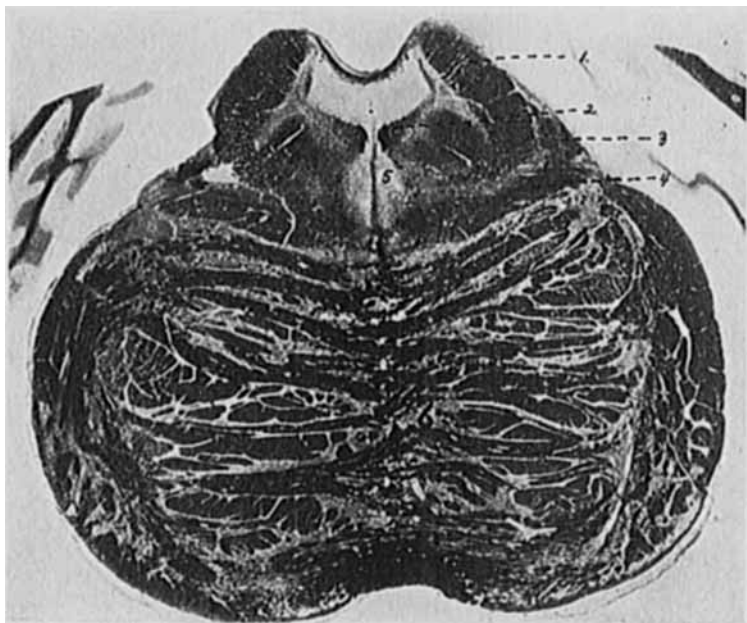nearly normal use of his hands and arms, and such improvement in the condition of his body and legs as to lead one to believe that if an unfortunate complication had not arisen, further improvement might have been expected. From a condition of total anæsthesia over the area above referred to, there was practically complete recovery by the ordinary tests.

Following death, a series of cross-sections of the spinal cord from the pons to the coccyx makes it possible to study the paths of degeneration following a crush at an unusual length of time after injury.

Pathological anatomy of the spinal cord shows that descending degeneration of all injured axones occurs in motor tracts below the level of the lesion and of the common tract, which is composed of short descending sensory collaterals from injured sensory neurones; it shows the occurrence of ascending degeneration of all sensory axones involved in the lesion.

The sections demonstrate that normal sensory and motor axones exist at the seat of the lesion, though their functions were interrupted at the time of injury, which suggested total destruction of the cord.

The operation showed fracture and depression of the spinous processes and laminæ, apparently lying against the cord. There was a blood-clot under the laminæ, none under the dura; condition of bodies of vertebræ not known.

CASE II.—Mr. M., twenty-seven years of age, dived from a raft into shallow water on July 20, 1898, and sustained an injury to his spinal cord. Dr. Samuel J. Mixter was called in consultation two days later, and advised immediate operation, which he performed. At the time of the operation there was diaphragmatic breathing, complete paralysis of motion and sensation below the level of the lesion; reflexes were absent; apparently a case of total transverse destruction of the cord.

The laminæ of the sixth and seventh cervical vertebræ were found fractured and depressed, lying against the spinal cord. These laminæ were removed, the dura was opened, and no macroscopic lesion of the cord detected. There were no blood-clots, wound closed, healing without interruption.

The patient was removed to the Massachusetts General Hospital on August 2, 1898; he was seen in consultation with Dr. H. C. Baldwin, and the following conditions recorded. Temperature, 101.4° F.; pulse, 96; respiration, 20. Both arms can be slowly flexed, but cannot be extended. The left leg can be moved very slightly, the right leg can be flexed about 30 degrees, tactile sensation diminished in both legs, reflexes present, the left patella reflex being increased; there is incontinence of urine and fæces.

August 8, 1898. Tactile sensation of right arm normal, except over ring and little finger; tactile sensation of left arm normal. The supinator longus muscle of both arms contracts, though no contraction can be felt in either triceps; both hands can be slightly flexed, with very slight extension of fingers. Some wasting of interossei muscles; incontinence of urine persists.

August 18, 1898. Condition has not changed much; he has a little freer motion of arms and of right leg; no improvement in the left leg.

August 23, 1898. Temperature, 103.4° F. Phlebitis of left leg and thigh.

September 10, 1898. Left ankle clonus present, with slight motion of toes; also slight motion in flexion of left knee.

September 11, 1898. Recovery from phlebitis.

October 3, 1898. Left arm; no motion in extension of forearm, no motion of fingers, flexion of wrist and forearm present, fair shoulder movements, pronation of forearm, but no supination. Right arm; extension of forearm to right angle, good motion of wrist and forearm, good shoulder movements. Left knee and ankle can be slightly flexed; there is contraction of left adductors, causing slight adduction but no abduction, no flexion of left thigh, but slight power of extension.

October 27, 1898. Patient lifted his left foot six inches from the bed. Right arm can be extended and moved freely. Less improvement in motion of left arm with scarcely any power of extension.

Discharged from the hospital, October 27, 1898.

November 2, 1898. A letter regarding his condition mentions " steady improvement at home; he can move his limbs quite well, can hold things in his hand, and even feed himself a little."

On July 17, 1900, a letter was written by the patient in a plain, legible hand stating that his " right side is much better than

the left side in every way. My right leg is as good as ever it was, every muscle as good 'though not as strong." " Right arm all right, except slight contraction, which is growing less. Right hand fairly good, thumb and forefinger nearly all right in regard to movement, but the sense of touch is not as acute as formerly. The other fingers of right hand not bad, but not quite as good. Still quite a contraction in left arm, triceps very weak, and did not seem to be good for anything until six months ago. Left hand not very good, fingers close when the wrist is straightened."

" Left leg; toe-drop still exists, no ankle motion, tendon contracted back of the heel. The cords in back of left knee are a little stiff, and are inclined to contract a little, though left leg can be straightened perfectly. General health excellent; can walk a half mile without resting, and am getting stronger. Took a trip of 200 miles with five changes without difficulty."

On April 3, 1901, the patient was examined by Dr. H. C. Baldwin, who kindly allows me to use the following notes:

" Patient's weight is 135 pounds. He now goes about with one cane. Deltoid muscles firm and of good strength. Slight wasting of left supra- and infraspinati muscles, but their movements are normal. Biceps muscles in good condition, and equally strong. The right triceps muscle is strong, the left one is feeble." Right arm can be fully extended, the left arm can be extended to an angle of 145 degrees, representing marked improvement in each. Complains of numbness in three ulnar fingers of right hand. There is considerable wasting of left forearm and hand, can flex the fingers of left hand moderately, but cannot separate fingers or use extensors of fingers. Supinator longus contracts. Disassociation symptoms, especially in the hands and over most of left arm. Knee-jerks are exaggerated, Babinsky reflexes present, left ankle clonus. The left leg is smaller and weaker than the right. Toe-drop present. Disassociation symptoms over both legs.

On December 2, 1903, the following notes were made by Dr. Baldwin, who gives permission to use them:

" The patient is engaged in insurance business and weighs 130 pounds. His condition has improved in every way; muscles are stronger, and can be used better. Can take off his collar and tie and put them on again, which is a distinct improvement.

" Tactile sensation in the thumb and forefinger of right hand is still absent.

"The shoulders are exceedingly well developed. The left triceps is stronger than at last visit, and the arm can be extended to 160 degrees. Condition of the legs same as at last visit."

The question is pertinent: What is the value of operation in injuries to the spinal cord?

1st. It removes depressed fragments of bone apparently lying against the cord; 2d, It removes blood-clots; 3d, Allows the escape of exudate and makes room for inflammatory thickening; 4th, If extensive hæmorrhage is present, either extra- or intradural, it relieves pressure from the cord. Cases in literature have shown that degeneration from pressure appears within four days; if a cord is injured by crush and not totally destroyed, the continued pressure of a blood-clot may succeed in completing total destruction. 5th, Traumatic spinal œdema may be of such extent as to demand greater space for enlargement of the cord to avoid further destruction of fibres; 6th, There is absolutely no method by which one can early diagnosticate slight or great pressure of a fragment of bone, the pressure of a small or a large hæmatoma, whether there is a momentary pinch of the cord or constant pressure; 7th, The fact that the cord looked normal in these cases does not preclude the possibility that pressure had existed, nor prove that a condition had existed in which drainage and relief of pressure were not distinctly beneficial; 8th, The patients did not suffer from any ill effect of the operation *per se,* the dangers of operation are very slight compared to the possible benefit to be derived therefrom; and the further satisfaction is obtained that the surgeon *knows* that continued pressure does not exist.

I feel justified in concluding, from the history and symptomatology, from the favorable progress, and from the study of the pathological anatomy in Case I, that operation is indicated in such injuries.

The statistics of fracture of the spine, contrasting operated and unoperated cases, the percentages of deaths, and average duration of life, have been concisely given in the *Journal of Nervous and Mental Disease,* 1902, Vol. xxix, by Dr. George L. Walton.

What observations can be drawn from these cases of unusual duration of life?

The symptoms of both cases were typical of a complete transverse lesion.

Kocher regards operation as out of the question in total transverse lesions. There was no sign to even suggest a partial transverse lesion, nor to suggest whether the cord had been momentarily pinched, or was being permanently pressed upon by blood-clot, laminæ, or fragments from the body of a vertebra, or by dislocation of vertebræ. All the material at hand was a completely paralyzed body with consciousness, slight motion in flexion of arms, and an unaided diaphragm.

Among the exhaustive methods of diagnosis, the X-rays may be invaluable, but too frequently are unsatisfactory.

There is one class of cases which has been reported, viz., extreme fracture of the vertebral bodies with extreme displacement, demonstrated by the Röntgen rays, in which it is wiser to resort to rectification and fixation,—the wisdom of this procedure being upheld in the report of a case.[12]

What, then, is the problem confronting the surgeon? Should the use of X-rays be unavailing in reaching a decision, the problem, without operation, results in one of speculation. I must use that word in the absence of more absolute information in regard to pressure of clot or bone upon the spinal cord.

If we start with the assumption that typical symptoms of a complete transverse lesion are to be accepted as infallible, the surgeon should not operate on such cases as these of which it is the object of this paper to report; but these cases prove as striking examples that typical symptoms of a complete transverse lesion are not infallible, in which case the surgeon is not doing all in his power to relieve the patient's condition unless he operates.

A case [13] is reported where there were classical symptoms of a complete transverse lesion in the region of the upper dorsal vertebræ. The arches of the three upper dorsal vertebræ were found depressed and impacted at operation and removed, followed by steady improvement.

How is one to decide whether a cord is crushed beyond repair or not? There are no symptoms which establish, otherwise than by their persistence, irremediable crush of the cord.

How long should a surgeon wait? Shock is the principal factor to be considered in this connection. As a rule, operation on most, if not all, of the cases can be delayed a few hours until a greater stability of the nervous system is regained. A certain few cases might impress one with the necessity of immediate operation.

It has been suggested by one operator (Kocher) that one may operate later when long-continued pressure is shown; but having waited that length of time, why is there, then, not a hesitation whether one has to do with pressure of a fragment, or a condition resulting from a momentary crush that has proved irremediable, and likewise stay the hand of the surgeon?

A case [14] is reported in which operation was delayed four days; marked degeneration was found at autopsy the fifth day. Why then delay for other than the constitutional condition—shock? I quote Dr. Walton: "We have no symptoms from which we can assert at the outset that the cord is crushed beyond at least a certain degree of repair, and that early operation in all doubtful cases will not only accomplish all that late operations would do, but also perform it before adhesions and permanent deformity exist by pressure from whatever cause."

Keen [15] is quoted as saying, if immediately after the accident the knee-jerks are absent and remain so, operation is contraindicated.

How long should one wait for this sign? and while waiting, may not pressure do irreparable injury? In Case I, knee-jerks did not return for twelve days; and it has been shown that degeneration may appear within four days after injury.

Case I, from its clinical aspect, was diagnosticated total transverse destruction of the cord at time of injury. Many of the fibres were not destroyed, as proved by the marked improvement in the symptoms, and also by microscopical examination, which a year after injury showed the presence of normal nerve-fibres in the degenerate area.

It seems reasonable, then, to assume that even with the appearance of total destruction we have to do with a condition which for a time has interrupted the registration of sensation and motion on the cerebrum over certain neurones, which after regaining equilibrium will resume their functions.

Though fibre degeneration has persisted through the year following the operation, the suggestion seems pertinent that the steady improvement indicates an increased transmission of impulses through the remaining scattered fibres; the analogue of which is found in the increase of functions occurring in the kidney after unilateral nephrectomy, showing the power of nature to accommodate herself to adverse conditions.

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